A Case Study of Gut Fermentation Syndrome (Auto-Brewery) with *Saccharomyces cerevisiae* as the Causative Organism

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**ABSTRACT**

Gut Fermentation Syndrome also known as Auto-Brewery Syndrome is a relatively unknown phenomenon in modern medicine. Very few articles have been written on the syndrome and most of them are anecdotal. This article presents a case study of a 61 years old male with a well documented case of Gut Fermentation Syndrome verified with glucose and carbohydrate challenges. Stool cultures demonstrated the causative organism as *Saccharomyces cerevisiae*. The patient was treated with antifungals and a low carbohydrate diet and the syndrome resolved.

Helicobacter pylori was also found and could have been a possible confounding variable although the symptoms resolved post-treatment of the *S. cerevisiae*.

**Keywords:** Auto-Brewery; Yeast; Fermentation; Gut Fermentation Syndrome

1. **Introduction**

Gut Fermentation Syndrome is a relatively unknown phenomenon in Western Medicine. This syndrome is difficult to research since it goes by several other names such as Auto-Brewery Syndrome and Endogenous Ethanol Fermentation. Most of the articles published on this syndrome are anecdotal. A few cases cite yeast as the causative agent; in particular *Candida albicans*, *Candida krusei*, *Candida glabrata*, and *Saccharomyces cerevisiae*. This article describes the case history of a 61 years old male from early symptoms, through diagnosis and treatment.

2. **Review of Literature**

Gut Fermentation Syndrome is described as a syndrome whereby patients become intoxicated without ingesting alcohol. In addition to the term Auto-Brewery, this syndrome has also been called Drunkenness Disease and Endogenous Ethanol Fermentation. The underlying mechanism is thought to be an overgrowth of yeast in the gut whereby the yeast ferments carbohydrates into ethanol. The earliest cases of this phenomenon were described in Japan. Iwata [1] detailed 12 cases prior to 1972.

In 1976, Kaji and others [2] described the case of a 24 years old female who became intoxicated after consuming carbohydrates which fermented in the gastrointestinal tract. In this situation the causative organisms were determined by cultures to be *Candida albicans* and *Candida krusei*. This patient restricted her intake of carbohydrates in the diet and received a course of an antifungal agent whereby all symptoms of her intoxication subsided.

Only a few cases have been reported in the last three decades. Two cases of particular note were identified in children. Dahshan and Donovan [3] described the case of a 13 years old girl with short gut syndrome who became intoxicated after ingesting carbohydrates. She had been placed in a rehabilitation facility with no access to alcohol. Aspirates from her small intestines grew *Candida glabrata* and *Saccharomyces cerevisiae*. After treatment with fluconazole, the symptoms resolved. The other case [4] was a 3 years old girl with short bowel syndrome who became intoxicated after ingesting a carbohydrate-rich fruit drink. Cultures from the gastric fluids demonstrated *Candida kefyr* and *Saccharomyces cerevisiae*. Again a course of fluconazole eliminated the symptoms.

Hunnisett and Howard [5] proposed a clinical test in
1990 to test for dietary fermentation. Subjects suspected of gut fermentation syndrome were given a fasting glucose challenge of 5 gm glucose orally. One gram of glucose was given in a hardened gelatin capsule to ensure passage into the duodenum. Blood glucose levels and blood ethanol levels were measured at 1 hour. Fasting blood alcohol levels were zero in nearly all subjects but 61% of the 510 subjects showed an increase in blood alcohol levels on the average of 2.5 mg/dl (range 1.0 - 7.0 mg/dl). This compared to near zero blood alcohol levels in the control group given the same challenge. Eaton [6] also summarized the phenomenon of gut fermentation with the above proposed diagnostic methods.

A study of 1557 residents of the United Arab Emirates [7] determined that fermentation of ethanol was “too low to have any forensic significance” (p. 149). However, it is important to note that the pooled maximum blood alcohol level was 3.52 mg/dl (0.035%) in males and 3.20 mg/dl (0.032%) in females. With zero tolerance of alcohol in the United Arab Emirates, there is no allowed legal limit.

More recently abnormal or unusual fermentation has been discussed in relation to high fiber diets [8], the use of ampicillin [9], and ingestion of prebiotic inulin [10]. Furthermore, Bivin and Heinen [11] conducted an experiment combining five infant food formulas and/or supplements with four common yeasts (C. albicans, C. tropicalis, Torulopsisglabrata, and S. cerevisiae) to measure ethanol production in vitro. All of the mixtures of yeast and carbohydrate produced ethanol with the S. cerevisiae preparations being the highest.

Saccharomyces cerevisiae, also known as brewer’s yeast, has a very well known history and life cycle because of the brewing industry. More recently the entire genome of S. cerevisiae has been identified due to its important use in fermenting corn into ethanol for fuel consumption. Very little is known, however, about the role of S. cerevisiae in humans. In fact, S. cerevisiae has been described only recently as an “emerging infectious disease” [12,13]. S. cerevisiae has mostly been identified as a pathogen in critically ill patients who are immuno-compromised [14,15] and in infants [16]. Not one single article could be found on an S. cerevisiae infection in an otherwise healthy, immuno-competent adult (Medline, 2011).

3. Case History

A 61 years old male, presented in January of 2010 with at least a five-year history of unexplained intoxication. In 2004, after surgery for a broken foot, and subsequent treatment with antibiotics, he began to seem excessively intoxicated after only two beers, and on occasion he would seem intoxicated without having been drinking. His wife, who is a nurse, began to document this phenomenon with a DOT approved alcohol breathalyzer. Often his blood alcohol percent was as high as 0.33 to 0.40. The legal limit for alcohol in the United States is 0.08 percent. They could find no correlation to these episodes other than scant ingestion of alcohol such as from a piece of gum with alcohol sugar or a candy with chocolate liqueur as an ingredient. The episodes were more frequent when a meal was missed, after exercise, or when alcohol had been ingested the night before.

The episodes of intoxication began to increase in severity and frequency over the ensuing years. In November of 2009, the subject was taken to the Emergency room on a day when he had not ingested alcohol. In the ER, his blood alcohol concentration was 371 (0.37%). He was admitted to the hospital for 24-hour observation and treated for severe alcohol intoxication. The physicians were not aware of any way that a person could be intoxicated without ingesting alcohol and therefore believed he must be a “closet drinker”.

In January of 2010, the patient presented to a gastroenterology practice where he underwent a complete gastroenterology workup. The patient had a history of hypertension and hyperlipidemia. His blood pressure was being treated but was not well controlled. All other systems were negative. The patient denied taking any type of yeast as nutritional supplementation such as probiotics and denied previous gastrointestinal disorders or treatments. Initially, routine breath tests were conducted for lactose and fructose intolerance as well as hydrogen and all were negative. A glucose tolerance was performed and was also negative. An EGD (esophagogastroduodenoscopy) and colonoscopy were conducted and were negative, however, Helicobacter pylori was isolated from his stomach. Stool cultures were also conducted that were positive for rare budding yeast and Saccharomyces cerevisiae.

In April of 2010 the patient was admitted to the hospital for a 24-hour observation period. His belongings were inspected to insure he did not have alcohol with him and no visitors were allowed during the 24-hour period. A glucose challenge was administered along with a high carbohydrate diet with snacks throughout the day. Blood was drawn for blood alcohol concentration (BAC) levels at baseline and every 2 hours and glucose levels every four hours. Breathalyzer levels were supervised by Texas DPS (Department of Public Safety) officers. At one point during the afternoon, the patient’s BAC rose to 120 mg/dl (0.12% per breathalyzer) in this controlled situation.

Unlike other case studies reported, fermentation in this case often occurred nearly 24 hours after the ingestion of sugar or alcohol.
4. Treatment

The patient was given an oral course of fluconazole (Diflucan) 100 mg a day for three weeks followed by a three week course of Nystatin 500,000 IU 4 times a day. He also took daily Acidophilus tablets to re-colonize the gut. During this six week period, the patient followed a very strict no sugar, no carbohydrate diet and did not ingest alcohol in any form. His breath alcohol level was tested frequently throughout each day and was 0.00 from the time treatment began until 10 weeks later. Stool cultures were then repeated and returned negative. Finally, the patient was treated with a course of Tetracycline for the H. pylori.

5. Conclusion

The authors believe this patient had Gut Fermentation Syndrome as documented informally by his wife and verified formally by the hospitalization glucose challenge and documentation of alcohol levels. The stool culture suggests that Saccharomyces cerevisiae was the causative agent and the fact that the stool cultures were negative for S. cerevisiae after treatment and the symptoms subsided at that time, supports this hypothesis. This is a rare syndrome but should be recognized because of the social implications such as loss of job, relationship difficulties, stigma, and even possible arrest and incarceration. It would behoove health care providers to listen more carefully to the intoxicated patient who denies ingesting alcohol. Gut Fermentation Syndrome warrants additional investigation to determine which organisms induce symptoms and what definitive tests should be conducted to confirm diagnosis. In addition, research would be important to determine how overgrowth occurs with S. cerevisiae when it is normally found as a commensal in the gut of humans.

REFERENCES


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